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Behavioral Specificity of β-Endorphin Suppression of Sexual Behavior: Differential Receptor Antagonism[1]

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WIESNER, J. B. AND R. L. MOSS. *Behavioral specificity of β-endorphin suppression of sexual behavior: Differential receptor antagonism.* PHARMACOL BIOCHEM BEHAV **24**(5) 1235–1239, 1986.—Open field behavior was observed in conjunction with mating behavior to discern whether the effect of intraventricular (ICV) β-endorphin (β-END) on sexual behavior may be secondary to akinesia. Three groups of ovariectomized, estrogen-progesterone-primed rats each received counterbalanced treatments of saline ICV, 2 μg β-END ICV, or 2 μg β-END ICV in combination with a selective opioid receptor antagonist. Receptive behavior (lordosis) and proceptive behaviors (presentation and ear wiggling) were consistently suppressed by β-END, while ambulation was unaffected. Rearing and grooming were generally decreased, although this effect was statistically significant in only one experiment. Pretreatment with the μ-1 antagonist naloxazone (50 mg/kg intravenously) reversed the effects of β-END on all behaviors tested. The δ receptor antagonist ICI-154,129 (12.5 and 50 μg ICV) only partially reversed the sexual effects of β-END but completely reversed the open field effects. It is concluded that the suppressive effect of β-END on sexual behavior, while not behaviorally specific, is not secondary to opioid-induced akinesia.

β-Endorphin Opioids Opiate receptors Lordosis Receptive behaviors Proceptive behavior Open field behavior μ Receptors δ Receptors

ENDOGENOUS opioid peptides have been implicated in the control of sexual behavior of male [5, 10, 11, 13, 18] and female [14, 20, 25, 26] rodents. The finding that opioids suppress sexual behavior in males has led various laboratories to conduct research to determine whether the effect is secondary to the cataleptic, akinetic, or nonspecific suppressant effects of opioids. Automated measures of activity [11, 13, 17] and feeding behavior [2] were unaffected by doses of opioids which suppressed sexual behavior, leading to the conclusion of behavioral specificity. However, certain other nonsexual behaviors were later reported to be suppressed by the sex-suppressant doses of opioids [10]. In ovariectomized (OVX) estrogen-progesterone (EP)-primed rats, intraventricular (ICV) administration of the opioid peptide beta-endorphin (β-END) has been shown to suppress lordosis behavior and certain proceptive behaviors [25, 26, 27]. We have recently studied the behavioral specificity of this effect and found it not to result from measurable catalepsy, general somatosensory defect, or alteration of blood pressure. Certain measures of open field activity were suppressed along with sexual behavior, indicating a lack of specificity, although the alteration of activity did not correlate with changes in sexual behavior, implying that the sexual effects may be independent of the activity effects of β-END [25]. As a means of determining whether the opioid effects are independent of one another, it was considered that they may be mediated by different opioid receptor types, and that selective antagonists could be instrumental in separating the behavioral effects of the opioid.

In the present study we have continued to examine the effects of β-END on sexual behavior in conjunction with open field behavior. These experiments have utilized preferential blockade of opioid receptor types in an attempt to dissociate the sexual effects from the open field effects, and to determine the receptor type(s) responsible for the opioid effects on behavior. The antagonists ICI-154,129 and naloxazone, used in these experiments, are known to be highly selective for δ receptors ([19]; S. J. Paterson, personal communication) and μ-1 receptors [15,28], respectively. The dose levels used have been found to be optimal for selective blockade of their respective receptor types *in vivo* [3, 7, 8, 22, 23, 24], and have been used to dissociate different endocrine effects of opioids [8, 22, 23]. The present studies indicate that the sexual effects of β-END occur independently of the open field effects, and may be mediated by multiple receptor types.

METHOD

Subjects

Sprague-Dawley female rats (200–300 g, Simonsen) were

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maintained in a temperature-controlled colony room with modified 14:10-hr light-dark cycle (fluorescent lights on 2200 hr, off 1200 hr) in which the dark phase was dimly illuminated by red light. Food and water were available ad lib. Under equithesin anesthesia, all animals were OVX and stereotactically implanted with permanent 23 gauge stainless steel cannulae directed into the third cerebral ventricle. At least one week after surgery, all animals were subjected to one or two preliminary mating tests in which experimental subjects were selected for their ability to respond to EP-priming by demonstrating high receptivity levels.

Steroid Priming

The priming protocol utilized pre-incubated, 15 mm Silastic capsules containing 17 β-estradiol [21], implanted subcutaneously (SC) 50–54 hr prior to testing. The animals received SC injection of progesterone (2.5 mg in oil) 4–6.5 hr prior to testing. Estrogen capsules were removed following each test session.

Sexual Behavior Tests

Mating tests consisted of placing the female in a semicircular 36×46 cm Plexiglas arena with two sexually active Long Evans males for 15 mounts; each mating test was completed within 15 minutes. The principal index of female receptivity was derived by dividing the number of lordoses by the number of mounts, to yield the lordosis-to-mount ratio (L/M). In addition, lordosis intensity was scored by a modified method of Hardy and DeBold [4]. Each lordosis was ranked from 1 to 3 (in increments of 0.5) according to the extent of dorsiflexion observed. For each test the numbers were totalled and divided by the number or lordoses scored, to yield a mean lordosis intensity (mean LI) for which the minimum was 1.0 and the maximum was 3.0. The proceptive behaviors of presentation ("posing") and ear wiggling were recorded and expressed as frequencies. These stereotypic behaviors have been described at length by Madlafousek and Hlinak [9]. Briefly, the presentation posture is a "freezing of the body with the pelvic area close to the floor and with the hind legs symmetrically spread sideways." Ear wiggling is a rapid vibration of the ears. The frequencies represent distinct instances of occurrence during the test session; this parameter is known to be correlated with sexual "heat" and is believed to indicate the propensity of the female to initiate copulation.

Open Field Tests

The open field arena consisted of four 18-inch high walls surrounding a square 40×40 in. wooden floor which was marked with a grid of sixteen 10×10 squares. The principal light source was a red 25 watt light bult suspended 20 inches above the center of the floor. The test was begun by placing an animal at the center of the floor, and its behavior was recorded for a period of 5 min. Behaviors scored, expressed as frequency of distinct instances during the test session, included crossings (crossing a line into an adjacent square), rearings (lifting front paws off the floor), grooming (a bout of washing, licking, scratching, biting, etc.), and defecation (number of fecal boli deposited). Following the open field test, the animal was placed in the mating arena for one minute prior to adding the males for subsequent testing of sexual behavior. The floor of the open field arena was sponged thoroughly between each test. The animals were subjected to preliminary open field tests in conjunction with the preliminary mating tests. Thus, pre-adaptation to the open field was roughly equal to that of the mating arena.

Drugs

Naloxazone, an irreversible μ-1 antagonist [15, 16, 28] and ICI-154,129 (ICI), a reversible δ-antagonist ([19]; S. J. Paterson, unpublished observations), were used in the same dose/time protocols found to be consistently effective and specific in previous endocrine experiments [8, 22, 23]. Naloxazone (a gift of Dr. J. I. Koenig, University of Chicago) was dissolved in 1% acetic acid; a dose of 50 mg/kg was injected intravenously (IV) by direct jugular puncture under ether anesthesia 25–30 hr prior to β-END infusion. Previous experiments have shown that this dose is effective for over 36 hr but for less than 7 days, by which time the bound receptors have presumably been replaced by a functional population. ICI (a gift of Dr. J. S. Shaw, ICI Pharmaceuticals) was dissolved in physiological saline and administered in a dose of 50 or 12.5 μg ICV in combination with β-END. β-END (Peninsula Laboratories) was dissolved in physiological saline and infused ICV in a 2 μg dose. Progesterone and 17 β-estradiol were purchased from Sigma.

Infusion and Test Procedure

ICV infusions and behavioral testing were conducted during the dark phase of the lighting cycle in low intensity red light within the colony room in which the animals were housed. For ICV infusions, animals were placed under brief (approximately 2.5 min) ether anesthesia. A 30 gauge inner cannula, connected with a Hamilton syringe by polyethylene tubing, was inserted into the permanent outer cannula; infusions (2 μl) were performed over 1 min, and the inner cannula was left in place for an additional minute. Thirty minutes after ICV infusion, each female was placed in the center of the open field arena. After a five minute recording period the animal was transferred to the mating arena for one minute prior to the addition of the males for the mating test. In each of the experiments, 10–13 EP-primed females were given three treatment combinations over three successive weeks in counterbalanced form. In experiment I, the treatments were vehicle IV + saline ICV, vehicle IV + β-END ICV, and naloxazone IV + β-END ICV (n=10). In experiment II, the treatments were saline, β-END, and β-END in combination with 50 μg ICI, all administered ICV (n=12). In experiment III, the treatments were saline, β-END, and β-END in combination with 12.5 μg ICI (n=13).

Statistical Analysis

Data were analyzed by repeated measures analysis of variance (ANOVA) followed by Newman-Keuls comparisons among the treatment groups.

RESULTS

Experiment I

β-END treatment did not significantly suppress any open field behaviors. However, as shown in Fig. 1, β-END significantly suppressed L/M ($p<0.025$). This effect on L/M was blocked by pretreatment with naloxazone ($p<0.025$, naloxazone/β-END vs. vehicle/β-END). Proceptivity scores showed typically high variance. Along with mean LI, proceptive behaviors appeared to be suppressed after β-END infusion although the effects were not significant; these effects were also apparently reversed by naloxazone,

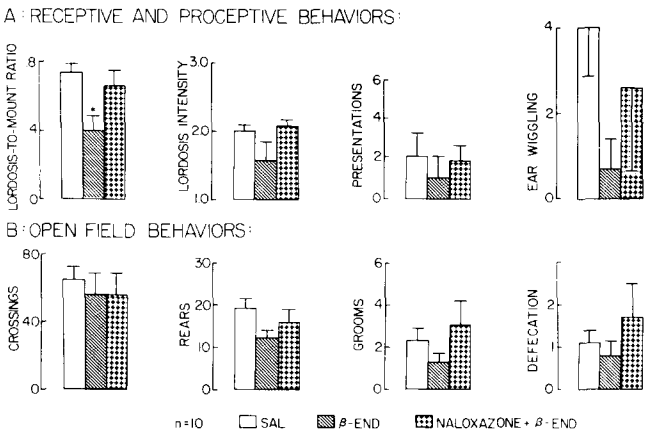

FIG. 1. Effects on sexual and open field behaviors of three treatment combinations: vehicle IV + saline ICV (SAL); vehicle IV + β-END ICV (β-END); or naloxazone IV + β-END ICV (naloxazone + β-END). Naloxazone/vehicle injections were administered at least 24 hours prior to ICV infusion. Naloxazone dose was 50 mg/kg; β-END dose was 2 μg (in 2 μl). Animals were OVX, EP-primed. *$p<0.025$ vs. saline score.

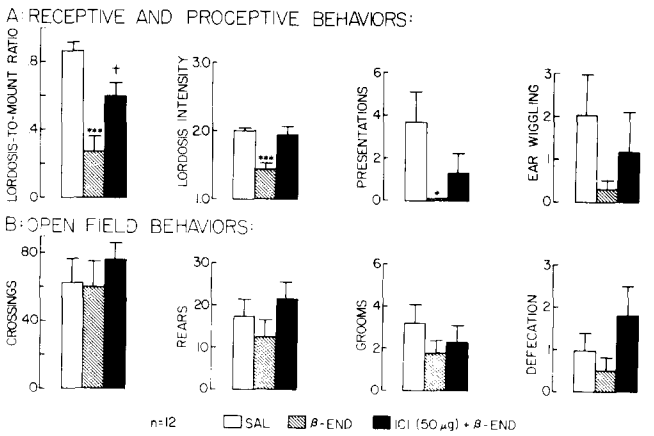

FIG. 2. Effects on sexual and open field behaviors of three treatments: 2 μl saline (SAL); 2 μg β-END (β-END); or 2 μg β-END + 50 μg ICI (ICI + β-END). Animals were OVX, EP-primed. *$p<0.025$, ***$p<0.005$, vs. saline score; †$p<0.005$, vs. both saline score and β-END score.

and naloxazone pretreatment significantly ($p<0.05$) increased mean LI over β-END scores. While none of the open field behaviors were significantly diminished by β-END, there was a tendency for rearing, grooming and defecation to be slightly diminished. Naloxazone pretreatment appeared to reverse any tendency of β-END to decrease these open field behaviors.

Experiment II

β-END treatment again had no significant effect on open field behaviors (Fig. 2). In contrast, β-END treatment significantly decreased L/M ($p<0.001$), mean LI ($p<0.005$), and presentations ($p<0.025$), and greatly decreased ear wiggling (non-significant). ICI (50 μg) only partially blocked each of these effects. The L/M scores after ICI/β-END infusion were significantly higher than after β-END alone ($p<0.005$) but significantly lower than saline control scores ($p<0.005$).

Suppression of mean LI by β-END was almost completely blocked by the 50 μg dose of ICI ($p<0.005$). There was a tendency for rearing, grooming, and defecation to be slightly decreased after β-END treatment; ICI interfered with those effects.

Experiment III

As shown in Fig. 3, β-END treatment significantly suppressed L/M ($p<0.001$) and mean LI ($p<0.005$), and abolished both ear wiggling ($p<0.05$) and presentations (non-significant). In addition, β-END treatment suppressed rearing ($p<0.01$) and grooming ($p<0.005$).

ICI (12.5 μg) again partially reversed the effect of β-END on mating behavior. L/M scores after ICI/β-END were significantly higher than after β-END alone ($p<0.01$) but significantly lower than after saline infusion ($p<0.05$). Reversal of other mating scores by this dose of ICI was not significant; both mean LI and ear wiggling were significantly lower after ICI/β-END than after saline infusion ($p<0.05$). In contrast, the suppressive effects of β-END on rearing and grooming are completely blocked by ICI: the ICI/β-END scores were significantly greater than the β-END scores ($p<0.005$), and

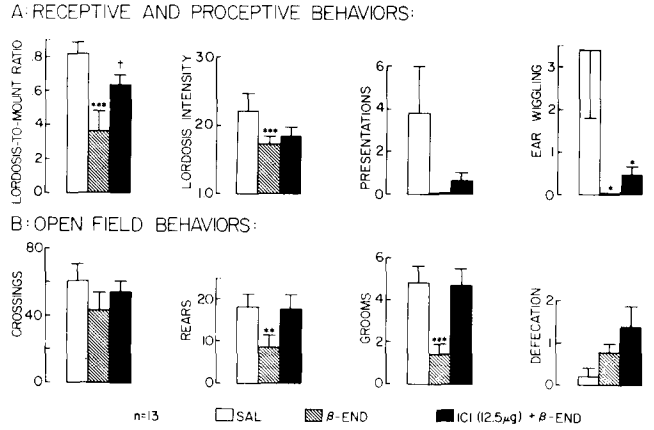

FIG. 3. Effects on sexual and open field behaviors of three treatments: 2 μl saline (SAL); 2 μg β-END (β-END); or 2 μg β-END + 12.5 μg ICI (ICI + β-END). Animals were OVX, EP-primed. *$p<0.05$, **$p<0.01$, ***$p<0.005$, vs. saline score; †$p<0.05$, vs. saline score and β-END score.

were virtually identical to the saline scores. Thus, ICI exerted a relatively selective antagonism of the open field effects of β-END as compared with the sexual effects of β-END.

DISCUSSION

Experiments described in this study have shown that: (1) the effects of β-END on open field behavior are less pronounced than on mating behavior and the behaviors appear to be altered independently; (2) ambulatory scores are less sensitive to β-END treatment than are some other open field behaviors (e.g., rearing), and are not suppressed by β-END under the conditions tested here; (3) the suppression of mating behavior (receptivity and proceptivity) by β-END is reversible by the μ-1 receptor antagonist naloxazone and partially reversible by the δ receptor antagonist ICI; (4) the

suppression of open field behavior by β-END appears to be completely reversed by both ICI and naloxazone. These experiments also serve to confirm previous results [27] demonstrating that non-cataleptic doses of β-END consistently suppress proceptive behaviors (presentations and ear wiggling) as well as receptive behavior (lordosis).

When the present experiments are considered together, the overall effects of β-END are very consistent and quite clear despite some variations in levels of significance. Primarily, L/M is significantly suppressed in all three experiments. Further, the profound suppression of proceptive behaviors is clear, with the exception of presentations in experiment I, in spite of the high variances which often preclude statistical significance using the ANOVA. (*Ad hoc* analysis using logarithmic transformation of this data indicates that β-END suppression of the proceptive behaviors was indeed significant in all cases.) While the suppression of lordosis intensity was not as robust, mean LI was reduced by 36% to 61% in all three experiments and lacked statistical significance in only one experiment. Overall, open field behavior was less effected by β-END than was sexual behavior. There was little tendency for ambulation (crossings) to be decreased. Rearing and grooming, however, were consistently lower after β-END treatment than after saline treatment, and this effect was statistically significant in experiment 3. Thus, the dose of β-END used in these experiments was less than that required to consistently exert significant suppression of the open field behaviors, even though it was adequate to significantly suppress receptivity in all cases.

The lack of effect of β-END on ambulation in the present experiments is in agreement with findings of other investigators that ambulation was unaltered after treatment with sex-suppressant doses of opioids [11, 13, 17]. However, the tendency for rearing to be decreased is consistent with findings of Meyerson [10] that certain behaviors other than ambulation, particularly rearing, were decreased by β-END in male rats. It is also consistent with findings of Browne and Segal [1] who found that β-END decreased rearing more than ambulation. It therefore appears that β-END suppression of sexual behavior may not be as specific as originally indicated by studies measuring ambulation with automated activity meters [11, 13, 17]. Rather, behaviors other than ambulation, particularly rearing, may be altered along with sexual behavior albeit to a lesser extent.

Results of a previous study [27] included findings that β-END-induced changes in mating behavior were not correlated with changes in open field behavior. This lack of correlation may reflect an independent action of β-END on the two types of behaviors, i.e., the effect on sexual behavior is not dependent on or secondary to an effect on motor activity. Results presented in the present report support that hypothesis, in that open field behaviors were usually altered to a lesser extent than were mating behaviors. Thus, while β-END suppression of sexual behavior is not necessarily 'specific,' it is not a result of akinesia.

Both ICI ([19]; S. J. Paterson, unpublished observations) and naloxazone [15,28] have been shown by various methods to be highly selective for δ and μ-1 receptors, respectively. The dose levels used in the present study have been found to be optimal for selective blockade of their respective receptor types *in vivo* [3, 7, 8, 22, 23, 24]. It is evident from the data of the present experiments that suppression of behavior by β-END can be mediated via both μ-1 and δ receptors. Suppression of sexual behavior was almost completely blocked by naloxazone, but only partially blocked by 50 μg ICI. These antagonistic effects of naloxazone and ICI contrast with their effects on β-END suppression of luteinizing hormone release [23], attesting to their pharmacological specificity. The 12.5 μg dose of ICI confirmed this specificity of effect as there was little decrement of the antagonistic action of ICI at the lower dose. Naloxazone, the more effective of the two antagonists, did not by itself alter the sexual behavior of estrogen-primed OVX rats when the antagonist was administered alone in a separate experiment (not shown).

Tendencies of β-END to suppress open field behaviors were almost totally blocked by both ICI and naloxazone. Although firm conclusions concerning receptor type involvement cannot be drawn due to the low activity of β-END on these behaviors, it would appear that δ receptors are involved more with these behaviors than with sexual behavior. In experiment III, in which two open field behaviors were significantly suppressed by β-END, a low dose of ICI completely antagonized the suppression of the open field behaviors while only moderately antagonizing the sexual behaviors. This relatively selective antagonism represents a partial dissociation between the effects of β-END on the two types of behavior, and supports the view that opioid effects on the two types of behavior are independent of each other. A more rigorous investigation of opioid receptor types involved in the β-END suppression of both sexual and open field behaviors awaits the availability of alternative antagonists.

It has been shown that lordosis behavior in the estrogen-primed female rat is facilitated by LHRH [12,18], and also that opioids are capable of suppressing release of LHRH into the hypophysial portal vessels [6]. It is tempting to speculate that β-END may suppress sexual behavior by suppressing release of LHRH at neural sites responsible for controlling lordosis. Research in this laboratory, however, has failed to support that hypothesis since LHRH, co-administered ICV with β-END, failed to reverse the suppression of lordisis by β-END (Wiesner and Moss, unpublished observations). These results are not consistent with those reported by Sirinathsinghji *et al.* [20] after administration of the peptides into the mesencephalic central gray. This discrepancy may be due to the differential routes of administration, although we have been unable to alter lordosis behavior by injecting β-END into the mesencephalic central gray in our experimental model. Clarification of this issue through further research will contribute greatly to the resolution of the problem of behavioral specificity of the opioid action.

In conclusion, it may be stated that suppression of female sexual behavior by β-END is not behaviorally 'specific,' since there is some tendency for certain non-sexual behaviors to decrease after β-END treatment at these doses. These changes could not be completely dissociated from sexual suppression by antagonizing different opioid receptor types, although a partial dissocation was exerted by antagonizing δ receptors. Reports of a specific action of β-END on male sexual behavior may have overlooked other measures of general activity besides ambulation. However, an important distinction must be made: lack of specificity does not imply that the effects on sexual behavior actually result from nonspecific akinetic effects. The lack of effect of β-END on ambulatory activity, the lack of correlation of sexual effects with open field effects, and the dissociating action of the δ antagonist suggest at least two possibilities: (a) ICV-administered β-END may influence neuronal pathways specifically involved with sexual behavior, while at the same time independently influencing pathways controlling certain other activities; or (b) ICV-administered β-END may exert

an effect on a higher function, such as arousal or affect, which manifests itself diffusely and variably among many but not all behaviors.

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